

VU Research Portal

A general enhancement of autonomic and cortisol responses during social evaluative threat

van den Bosch, J.A.; de Geus, E.J.C.; Carroll, D.; Goedhart, A.D.; Anane, L.A.; van Zanten, J.J.; Helmerhorst, E.J.; Edwards, K.M.

published in

Psychosomatic Medicine
2009

DOI (link to publisher)

[10.1097/PSY.0b013e3181baef05](https://doi.org/10.1097/PSY.0b013e3181baef05)

[Link to publication in VU Research Portal](#)

citation for published version (APA)

van den Bosch, J. A., de Geus, E. J. C., Carroll, D., Goedhart, A. D., Anane, L. A., van Zanten, J. J., Helmerhorst, E. J., & Edwards, K. M. (2009). A general enhancement of autonomic and cortisol responses during social evaluative threat. *Psychosomatic Medicine*, 71(8), 877-885.
<https://doi.org/10.1097/PSY.0b013e3181baef05>

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal ?

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

E-mail address:

vuresearchportal.ub@vu.nl

A General Enhancement of Autonomic and Cortisol Responses During Social Evaluative Threat

JOS A. BOSCH, PhD, ECO J. C. DE GEUS, PhD, DOUGLAS CARROLL, PhD, ANNEBET D. GOEDHART, MA,
LEILA A. ANANE, MSc, JET J. VELDHUIZEN VAN ZANTEN, PhD, EVA J. HELMERHORST, PhD, AND KATE M. EDWARDS, PhD

Objective: To examine the Social Self Preservation Theory, which predicts that stressors involving social evaluative threat (SET) characteristically activate the hypothalamic-pituitary-adrenal (HPA) axis. The idea that distinct psychosocial factors may underlie specific patterns of neuroendocrine stress responses has been a topic of recurrent debate. **Methods:** Sixty-one healthy university students ($n = 31$ females) performed a challenging speech task in one of three conditions that aimed to impose increasing levels of SET: performing the task alone (no social evaluation), with one evaluating observer, or with four evaluating observers. Indices of sympathetic (preejection period) and parasympathetic (heart rate variability) cardiac drive were obtained by impedance- and electrocardiography. Salivary cortisol was used to index HPA activity. Questionnaires assessed affective responses. **Results:** Affective responses (shame/embarrassment, anxiety, negative affect, and self-esteem), cortisol, heart rate, sympathetic and parasympathetic activation all differentiated evaluative from nonevaluative task conditions ($p < .001$). The largest effect sizes were observed for cardiac autonomic responses. Physiological reactivity increased in parallel with increasing audience size ($p < .001$). An increase in cortisol was predicted by sympathetic activation during the task ($p < .001$), but not by affective responses. **Conclusion:** It would seem that SET determines the magnitude, rather than the pattern, of physiological activation. This potential to perturb broadly multiple physiological systems may help explain why social stress has been associated with a range of health outcomes. We propose a threshold-activation model as a physiological explanation for why engaging stressors, such as those involving social evaluation or uncontrollability, may seem to induce selectively cortisol release. **Key words:** social evaluation, autonomic reactivity, HPA-axis, response specificity, shame, self-esteem, psychological stress.

ECG = electrocardiograph; HPA = hypothalamic-pituitary-adrenal; ICG = impedance cardiograph; PEP = preejection period; RMSSD = root mean square of successive differences; SAM = sympathetic-adrenal-medullary; SET = social evaluative threat; TSST = Trier Social Stress Test.

INTRODUCTION

Response specificity, the idea that particular characteristics of a stimulus or an individual are associated with distinct neuroendocrine and physiological response patterns, is one of the fundamental assumptions in psychophysiology and a dominant hypothesis in biobehavioral medicine (1–5). To most stress researchers this notion is intuitively obvious, buttressed by the evolutionary argument that different physiological response patterns are required to cope adaptively with different threats (6). Response specificity may also explain why certain types of stress have a more profound health impact than others, or why some stressors seem associated with specific pathologies (4,7,8).

Given the central role of the hypothalamic-pituitary-adrenal (HPA) axis in the stress response and its important physiological functions (9,10), substantial research has been di-

rected at identifying the psychological determinants of its activation (11,12). A guiding, but sometimes implicit, assumption is that the psychological determinants of HPA activation can be distinguished from those that activate other stress-response systems (e.g., sympathetic-adrenal-medullary (SAM) system or parasympathetic nervous system) (7,11,13,14). For example, it has been proposed that stressors involving novelty, lack of control, or loss/harm-appraisals preferentially activate the HPA axis whereas factors like effort, arousal, or challenge-appraisals drive SAM activation (7,11,13–15). However, the status of such models does not always seem to be matched by the strength of their empirical support; much of the supportive evidence takes the form of extrapolations from nonhuman studies, and the human data remain inconclusive (11). It is perhaps hardly surprising, then, that the specific psychological determinants of HPA activation continue to be a topic of research and debate.

The Social Self Preservation Theory is the most recent contribution to this debate (16,17). This theory, formulated by Gruenewald and co-workers, predicts that “. . . threats to the social self, or situations which threaten to demean one’s social image or standing, engender a specific set of psychological and physiological reactions” (17). These specific reactions are proposed to be feelings of low social worth, accompanied by self-conscious emotions, such as embarrassment and shame and, in the physiological domain, increases in HPA activation (16,17). Key support for the theory was adduced from a meta-analytic review of 208 acute laboratory stress studies, which demonstrated that stress exposure paradigms characterized by social-evaluative threat, such as stressful situations in which an evaluative audience was present or in which the participant was the target of a negative social comparison, led to greater cortisol reactivity than paradigms in which social-evaluative threat was absent or minimal (11). The authors concluded that this meta-analysis supported a “stressor-physiology specificity perspective” (11).

From the School of Sport and Exercise Sciences (J.A.B., D.C., L.A.A., J.J.V.v.Z., K.M.E.), University of Birmingham, United Kingdom; Mannheim Institute of Public Health, Social, and Preventive Medicine (J.A.B.), Mannheim Medical Faculty, University of Heidelberg, Germany; Department of Biological Psychology (E.J.C.d.G., A.D.G.), Vrije University, Amsterdam, Netherlands; Department of Periodontology and Oral Biology (E.J.H.), Goldman School of Dental Medicine, Boston University, Boston, Massachusetts; and the Department of Psychiatry (K.M.E.), University of California San Diego, San Diego, California.

Address correspondence and reprint requests to Jos Bosch, School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, B15 2TT, UK. E-mail: j.a.bosch@bham.ac.uk

Received for publication November 5, 2008; revision received June 3, 2009.

This study was funded, in part, by the Birmingham Interdisciplinary Bridging in Engineering, Medicine and Science and National Institutes of Health Grant RO3-DE-16726 from the National Institute of Dental and Craniofacial Research (J.A.B.).

DOI: 10.1097/PSY.0b013e3181baef05

Subsequent empirical support for this specificity perspective was provided by Gruenewald and colleagues (17). In this study, participants underwent the Trier Social Stress Test (TSST) protocol (18), which involves delivering an impromptu speech and performing a mental arithmetic task, in the presence or absence of evaluative others. As predicted by the Social Self Preservation Theory, performing the TSST in the presence of an evaluative audience provoked larger increases in self-reported shame/embarrassment than performing the task alone. Further, cortisol increased only when the TSST was performed with the audience present, whereas the two task conditions elicited largely similar increases in heart rate and blood pressure. These findings suggest that the SAM system responds less sensitive to social evaluative threat than the HPA axis, reinforcing the view that there may be distinctive psychological determinants of HPA activation. At the same time, however, the observation that cardiovascular and autonomic indices minimally differentiate between evaluative and nonevaluative conditions conflicts with the general finding in social facilitation and social anxiety research (19,20). Such research has shown that tasks involving high evaluative threat (for example, caused by the presence of an observer, the presence of a high status observer, or because of being evaluated on valued traits like intelligence) provokes much larger cardiovascular and autonomic reactions than situations of low evaluative threat (19,21,22). Indeed, evaluated performance tasks, such as speech performance, have become routinely employed in stress research exactly because of their robust activation of the autonomic and cardiovascular system (23–25).

Considering the theoretical importance of the study by Gruenewald et al. (17), providing the strongest empirical support for the Social Self Preservation Theory to date, replication using a more detailed assessment of autonomic nervous system activity seemed appropriate. Two additional major modifications were made to their original study protocol. First, we noted that the TSST protocol utilized by Gruenewald and co-workers required participants to change repeatedly between sitting and standing posture and to walk between different areas of the laboratory (17). Whereas cortisol release is relatively insensitive to modest physical activity (26), movement and posture changes substantially affect cardiovascular and autonomic measures (27–31). Evidence confirms that somatic activity confounds cardiovascular and autonomic responses during the TSST (32). To prevent such confounding, we controlled movement by keeping study participants in the same position (sitting) throughout the pretask baseline and all experimental procedures. Second, although the present study similarly manipulated social evaluative threat by performing a demanding task (impromptu speech delivery) alone or in front of an evaluating audience, we included two different audience sizes (one observer or four observers) to manipulate levels of social evaluation (33).

Three specific hypotheses were tested. First, on the basis of the findings by Gruenewald et al. (17), it was expected that social evaluation would enhance cortisol reactivity but not, or

only modestly, autonomic and cardiovascular reactivity. Second, on the same basis, we further expected that cortisol reactivity, but not autonomic and cardiovascular activity, would increase in parallel with the degree of social evaluative threat, i.e., show a dose-dependent relationship with audience size (33). Third, to further test the specificity of HPA responses during evaluative threat, it was predicted that HPA activity would increase independently of autonomic and cardiovascular responses but would correlate with increases in shame/embarrassment.

METHODS

Participants

Sixty-one university undergraduates ($n = 31$ women) participated in the present study (mean \pm standard deviation age = 20.3 ± 1.09 years; range = 18–24 years). Participants were recruited via advertisements in lecture rooms and by posters on campus, and were given course credit hours or paid £5 for completion of the study. Exclusion criteria were a) suffering from an immune, cardiovascular, metabolic, or kidney disorder; b) current cold or respiratory infection; c) use of prescribed medication (excluding the contraceptive pill) during the previous month; d) pregnancy or suspected pregnancy; e) being a smoker. Participants were instructed not to consume food or caffeinated beverages 2 hours before testing and to abstain from alcohol and strenuous exercise 12 hours before testing. Based on research showing that females in the follicular phase and those taking oral contraception show comparable cortisol responses to stress and adrenocorticotrophic hormone infusion (34), female participants were tested between days 4 to 7 post menses, or, when oral contraceptives were used, on a day the pill was taken. The study was approved by the ethical review committee of the School of Sport Exercise Sciences. Data were collected between November 2005 and March 2006.

Procedures

Task Preparations

Participants were invited to complete a single 2-hour afternoon testing session, commencing between 1 PM and 4 PM. On arrival at the laboratory, informed consent was obtained and electrodes for impedance cardiography (ICG) and electrocardiography (ECG) were attached. Participants were seated comfortably in a chair with arm rests, and were instructed to maintain a similar posture throughout the session and minimize movement. The latter was facilitated by the use of supportive pillows, and by the positioning of legs in a box, which did not physically restrict movement but limited the perimeter for movement. Participants were then provided with a standardized snack (1178 kJ; 43.1 g carbohydrate, comprising 26.3 g glucose; 10.9 g fat; 2.6 g protein) and a glass of water (250 mL). During the subsequent 45 minutes baseline period, participants completed a set of questionnaires and engaged in quiet reading. During the final 6 minutes of this baseline, at the same time still engaged in quiet reading, cardiac activity was assessed unobtrusively. At the completion of this baseline measurement, the first saliva sample (“baseline”) was taken, and a second set of questionnaires was administered. Subsequently, the task was explained and initiated.

Stress Task

The task consisted of two back-to-back speeches, each with 2 minutes of preparation and 4 minutes of speech delivery (35,36). For the first speech, participants were required to argue convincingly they were wrongly accused of shoplifting (37). For the second speech task, participants were asked to reveal and explain three of their best and three of their worst characteristics (38). The total task duration was 15 minutes, including instructions. Task instructions and timing were standardized by use of a DVD which was played on a TV screen. The experimenter left the room after starting the DVD and remained outside during the task. Participants were informed that the experimenter could still be contacted through a two-way radio.

RESPONSES TO SOCIAL EVALUATIVE THREAT

Conditions

Participants were allocated randomly to one of three conditions, structured to provoke increasing social evaluative threat: 1) a nonevaluative or no-audience condition during which nobody was present as the participant undertook the speech tasks; 2) a social evaluation condition whereby a single audience member (of opposite sex) was present; and 3) a social evaluation condition whereby four people (two men and two women) were present. Participants in the social evaluative conditions were told that the audience would be evaluating their speech performance and ability to communicate ideas successfully in a social situation. Participants in the nonevaluative condition were informed that they would perform the task alone in the room and that their performance was under no form of evaluation. The two-way radio was merely used to check if the speeches were on the correct topic (all participants followed speech instructions). The specifics of the task condition were only revealed at the point when task preparations were initiated to avoid baseline differences in anticipatory arousal. After the task was described, audience members entered the room (in the social evaluative conditions) and were seated in front of the participant. Audience members were approximately the same age as the participants and trained to adopt a nonaccepting and critical manner as described by Gruenewald and associates (17). If the participants stopped speaking for a period exceeding 20 seconds, the experimenter would prompt by sounding an alert through the two-way radio. This procedure was to ensure that the participants spoke for the full period in all three conditions.

Immediately after completion of the speech tasks, the experimenter reentered the room, collected the second saliva sample, and issued the third set of questionnaires. At the same time, the audience left the room. During the subsequent 45-minute recovery period, a saliva sample and mood questionnaire data were obtained at 15-minute intervals.

Questionnaires

Health behaviors were assessed by questionnaire and included assessments of exercise, alcohol and caffeine consumption, sleep, health complaints, use of nonprescribed medication, and menstrual cycle phase. The Test Anxiety Scale (39) is a 37-item ("true/false") questionnaire that assesses test anxiety as a situation-specific personality trait ($\alpha = 0.82$); the Fear of Negative Evaluation scale (40) is a 12-item questionnaire (4-point Likert scale; 1 = Very little to 4 = Much) that provides an index of social anxiety ($\alpha = 0.94$). The State Self-Esteem Scale (41) was administered pre and post task. For this 20-item scale, respondents had to rate their current thoughts regarding confidence, social self-esteem, and performance on a 5-point Likert scale (1 = Not at all to 5 = Extremely). For the current study, only the performance and social subscales were used (Performance $\alpha_{pre} = 0.82$, $\alpha_{post} = 0.88$; Social $\alpha_{pre} = 0.86$, $\alpha_{post} = 0.91$). At both pretask and posttask time points, participants were also administered an extended version of the Affect Balance Scale (42), which is a 43-item (Likert format; 1 = Not at all to 5 = Very strong) measure of positive and negative affect. Participants rated the emotions experienced over "the preceding minutes" (baseline, recovery) or "during the task." For the present study, we analyzed the negative affect subscale anxiety subscale (e.g., nervous, timid, anxious; $\alpha_{pre} = 0.78$, $\alpha_{post} = 0.85$), and the shame/embarrassment subscale¹ (items: embarrassed, self-conscious, ashamed, humiliated; $\alpha_{pre} = 0.74$, $\alpha_{post} = 0.87$). The latter subscale is an extension of the Affect Balance Scale developed by Gruenewald and associates (17). The pre- and posttask questionnaires were supplemented by seven single-item questions (using a 7-point Likert scale) assessing difficulty, stressfulness, arousal, performance, embarrassment, confusion, and engagement. Pretask items were formulated to assess task expectations (e.g., "How difficult do you expect to find the task?").

Cardiac and Autonomic Measures

Assessment of cardiac responses focused on cardiac sympathetic and parasympathetic control (35,43,44). Indices of sympathetic and vagal drive

¹This subscale has been developed as a measure of "shame" for the study by Gruenewald and colleagues (17). Because the scale contains both embarrassment and shame items, it was denoted as "shame/embarrassment" in this paper.

were obtained through analyses of ECG and thoracic ICG signals. Signals were recorded continuously throughout the experiment with six Ag/AgCl spot electrodes (AMI type 1650-005, Medtronic, Minneapolis, Minnesota), using the Vrije Universiteit Ambulatory Monitoring Device (VU-AMD, Vrije Universiteit, Amsterdam, Netherlands) (45). ECG and ICG complexes were ensemble averaged with reference to the ECG R wave across 1-minute time points. From these 1-minute ensembles, average levels were computed for heart rate (HR), preejection period (PEP), the Root Mean Square of Successive Differences (RMSSD), respiratory frequency, and a respiratory depth (tidal volume). These minute-by-minute means were averaged over the 6-minute pretask baseline, each 6-minute stressor (2 minutes preparation plus 4 minutes speech), and a 6-minute recovery (15-minute post task) (36,46). PEP was used as an index of cardiac sympathetic drive, and RMSSD as a measure of cardiac parasympathetic activity (47,48).

Saliva Collection and Cortisol Assessment

Saliva was collected using Salivettes (Sartstead, Oxford, UK). For each collection, participants were instructed to place the Salivette under the tongue for 3 minutes and not to chew. Once the 3 minutes had elapsed, subjects returned the Salivette into a sealed plastic tube. The samples were centrifuged at room temperature for 5 minutes at 3000 g, and saliva was divided into 500- μ L aliquots and frozen at -20°C until assayed. Salivary cortisol was measured using a competitive enzyme-linked immunosorbent assay, and analysis was carried out (sensitivity = <0.7 ng/mL, intra-assay variability = 9.2%) according to the manufacturers' instructions (R&D Systems, Minneapolis, Minnesota).

Statistical Analysis

An initial comparison of baseline differences was performed using a series of univariate analyses of variance (ANOVAs). The psychological and physiological responses were examined using repeated-measures multivariate analyses of variance (MANOVAs), which treated the different sampling times (pretask, posttask for psychological variables; baseline, task 1, task 2, recovery for cardiovascular and autonomic variables; baseline, posttask, +15 minutes, +30 minutes, and +45 minutes recovery for cortisol) as a within-subject factor and the condition as a between-subject factor. Separate Time \times Condition ANOVAs compared the responses during each condition in a pairwise fashion. Two subjects had baseline cortisol levels >3.5 standard deviation above the mean, and were excluded from the analyses. Eta-squared (η^2) is reported as a measure of effect size. Heart rate variability (RMSSD) was log transformed [$\ln(\text{RMSSD} + 1)$] for statistical analyses and also presented in the figures. Occasional missing data are reflected in the slight variations in degrees of freedom. Data were analyzed using SPSS 15.01 for Windows (SPSS, Chicago, Illinois).

RESULTS

Group Differences

There were no differences among the three experimental groups in age, body mass index (BMI), or ethnicity (MANOVA $F(2,58) = 1.43$, $p = .25$, $\eta^2 = 0.037$). Trait Fear of Negative Evaluation and Anxiety were also similar between groups (MANOVA $F(2,58) = 2.40$, $p = .11$, $\eta^2 = 0.046$). As can be seen in Table 1, at baseline no significant group differences were observed in the affective state (state anxiety, shame/embarrassment, social self-esteem, total negative affect) or task expectations (difficult, stressful, arousing, confusing, engaging, or embarrassing). Similarly, no baseline group differences were observed for physiological measures (namely, HR, PEP, RMSSD) or cortisol (MANOVA $F(2,58) = 0.56$, $p = .576$, $\eta^2 = 0.019$). The number of participants who required prompting to continue speaking after a 20-second silence was approximately similar for each condition (3, 2, and 3 for the 0-, 1- and 4-audience conditions, respectively).

Affective Responses

Figure 1 presents an overview of affective responses during the tasks. Repeated-measures ANOVA revealed significant effects of time for anxiety ($F(1,58) = 79.76, p < .001, \eta^2 = 0.579$), shame/embarrassment ($F(1,55) = 88.67, p < .001, \eta^2 = 0.617$), total negative affect ($F(1,57) = 36.88, p < .001, \eta^2 = 0.393$), and social self-esteem ($F(1,57) = 14.11, p < .001, \eta^2 = 0.198$), but not for performance self-esteem ($F(1,58) = 0.21, p = .645, \eta^2 = 0.004$). Significant Time \times

Table 1. Mean (SD) Baseline Values in Each Condition

	No Audience	1-Audience	4-Audience
Age	20.6 (1.4)	20.4 (1.0)	20.0 (0.9)
BMI	22.5 (2.2)	23.0 (2.6)	22.4 (1.7)
Fear of negative evaluation	25.8 (6.0)	29.6 (8.6)	24.8 (8.0)
Anxiety (ABS)	1.4 (0.5)	1.5 (0.6)	1.5 (0.5)
Shame/embarrassment (ABS)	1.4 (0.5)	1.6 (0.8)	1.5 (0.8)
Negative affect (ABS)	1.2 (0.2)	1.3 (0.3)	1.3 (0.4)
Social self-esteem	3.9 (0.5)	3.8 (0.6)	3.9 (0.7)
Task expectations			
Difficulty	1.7 (0.9)	1.7 (1.3)	1.8 (1.4)
Stressfulness	1.8 (1.2)	1.7 (1.3)	1.9 (1.3)
Arousing	1.6 (1.2)	1.7 (1.1)	1.9 (1.2)
Performance	3.0 (0.7)	3.4 (0.9)	3.3 (0.9)
Confusing	1.9 (0.9)	2.2 (1.2)	2.1 (0.9)
Engaging	2.7 (0.9)	3.5 (1.0)	3.1 (1.1)
Embarrassing	1.9 (1.3)	2.1 (1.5)	1.9 (1.7)

BMI = body mass index; ABS = Affect Balance Scale.

Condition interactions were found for anxiety ($F(2,58) = 3.96, p = .024, \eta^2 = 0.120$) and shame/embarrassment ($F(2, 55) = 3.26, p = .046, \eta^2 = 0.106$), and a marginal interaction effect emerged for total negative affect ($F(2, 57) = 2.90, p = .064, \eta^2 = 0.092$). Table 2 presents the results of subsequent pairwise analyses of Time \times Condition interactions (i.e., comparing affective responses in the no-audience with either the 1- or 4-audience conditions as well as comparing the two audience conditions). These analyses revealed significantly greater increases in anxiety, shame, and negative affect in the audience conditions in comparison with the no-audience condition; these affective responses did not differ between the two audience conditions (Table 2).

Physiological Responses

Cardiac and Autonomic Responses

Figure 2 presents the summary data of cardiac autonomic responses. Repeated-measures ANOVA yielded significant Time \times Condition interactions for HR ($F(6,162) = 5.45, p < .001, \eta^2 = 0.168$), PEP ($F(6,162) = 9.74, p < .001, \eta^2 = 0.265$), and RMSSD ($F(6,159) = 3.76, p = .002, \eta^2 = 0.124$). Subsequently, we again compared the responses for each condition in a pairwise fashion, using repeated-measures ANOVA (Table 3). These analyses demonstrated that, for HR and PEP, both the 1- and 4-audience conditions induced significantly larger responses than the control condition (Table 3). Further, the 4-audience condition elicited larger changes in HR and PEP than the 1-audience condition.

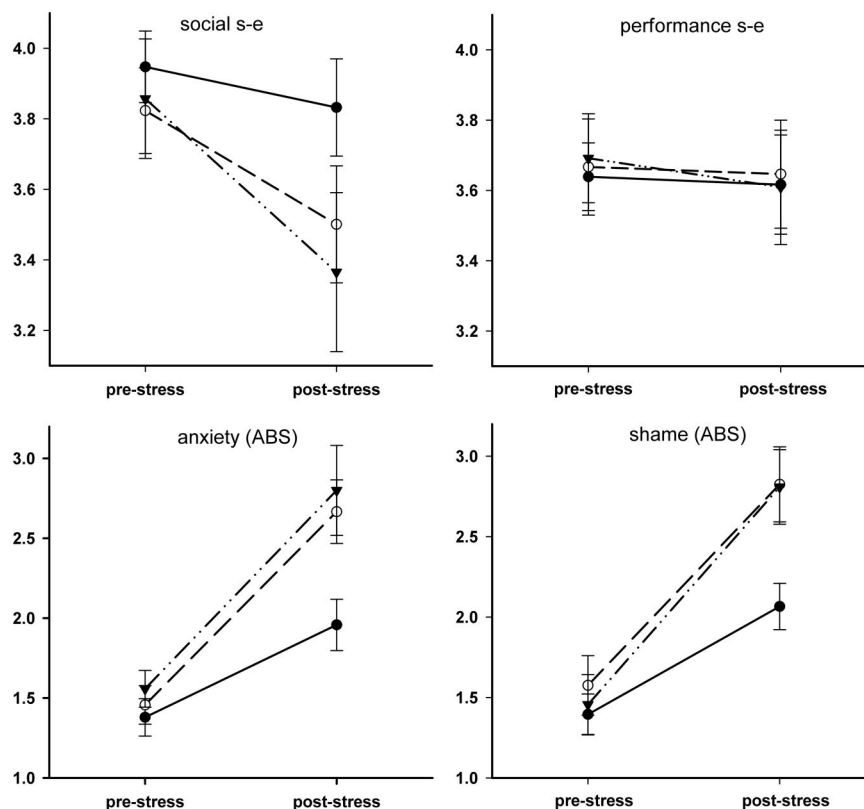


Figure 1. Mean \pm standard error of the mean values pre- and poststressor scores on self-esteem (*s-e*), anxiety and shame/embarrassment in the no-audience (filled diamonds), 1-audience (open circles), and 4-audience (filled triangles) conditions.

RESPONSES TO SOCIAL EVALUATIVE THREAT

TABLE 2. Univariate Statistical Analyses (Repeated-Measures Analysis of Variance) of Time by Condition Interactions

	Anxiety	Shame	Negative Affect
Control vs. 1-audience	$F(1,39) = 2.30$, $p < .01$, $\eta^2 = 0.172$	$F(1,38) = 5.16$, $p < .05$, $\eta^2 = 0.120$	$F(1,39) = 4.63$, $p < .05$, $\eta^2 = 0.106$
Control vs. 4-audience	$F(1,38) = 4.83$, $p < .05$, $\eta^2 = 0.113$	$F(1,36) = 4.80$, $p < .05$, $\eta^2 = 0.117$	$F(1,37) = 5.80$, $p < .05$, $\eta^2 = 0.135$
1-Audience vs. 4-audience	$F(1,39) = 0.01$, $p = .915$, $\eta^2 = 0.000$	$F(1,36) = 0.01$, $p = .886$, $\eta^2 = 0.001$	$F(1,38) = 0.07$, $p = .798$, $\eta^2 = 0.002$

Significant time by condition interactions are highlighted in bold.

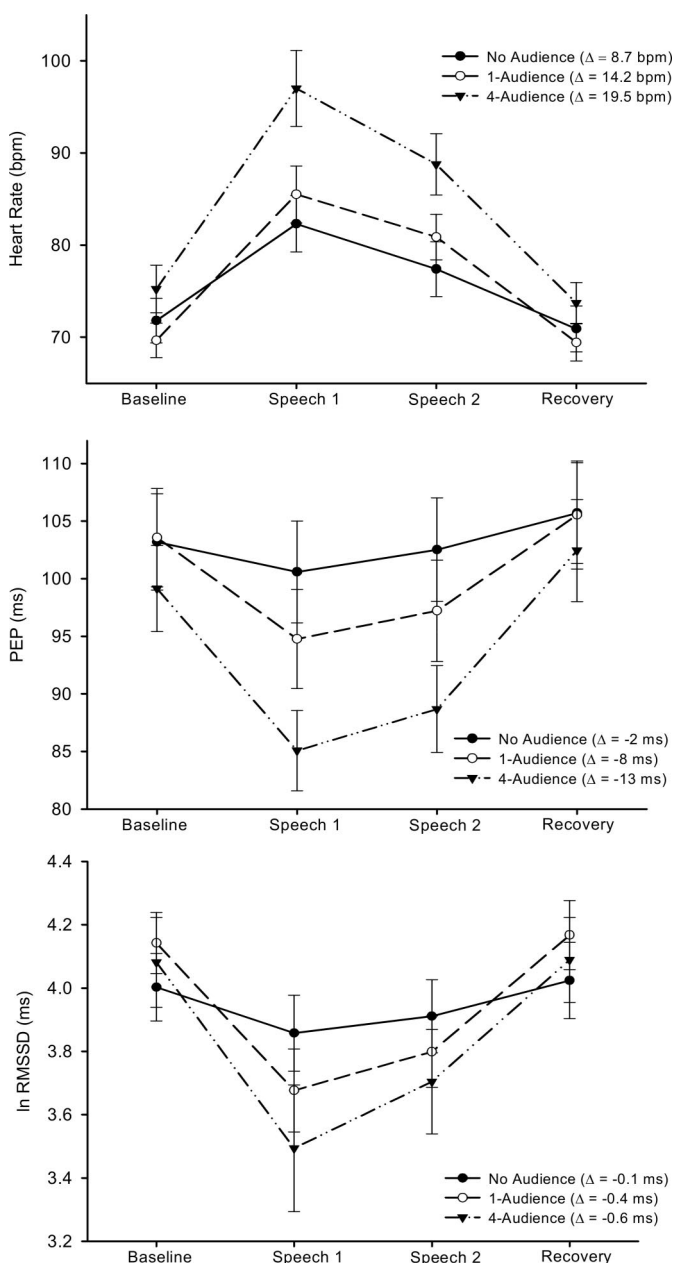


Figure 2. Mean \pm standard error of the mean values in the no-audience (filled circles), 1-audience (open circles), and 4-audience (filled triangles) stressor conditions across the session (Δ = speech values – baseline values). Presented are: heart rate (upper graph); preejection period (PEP) (middle graph); root mean square of successive differences (RMSSD) (lower graph).

RMSSD similarly showed larger responses in the 1- and 4-audience conditions than in the control condition, but responses to the 1- and 4-audience conditions were not significantly different (Table 3). Essentially the same outcomes emerged when these analyses were repeated, using only the 2-minute preparation period, to exclude respiration artifacts caused by speaking. We also analyzed respiratory patterns to further determine if the effects on RMSSD could have been confounded by concomitant condition effects on respiratory frequency and tidal volume (the 2-minute no-speech preparation period was used for these analyses). However, no significant Time \times Condition interactions emerged for respiration (data not shown). The effects of condition on HR, PEP, and RMSSD remained unaltered after adjustment for baseline values, gender, and BMI.

Salivary Cortisol Responses

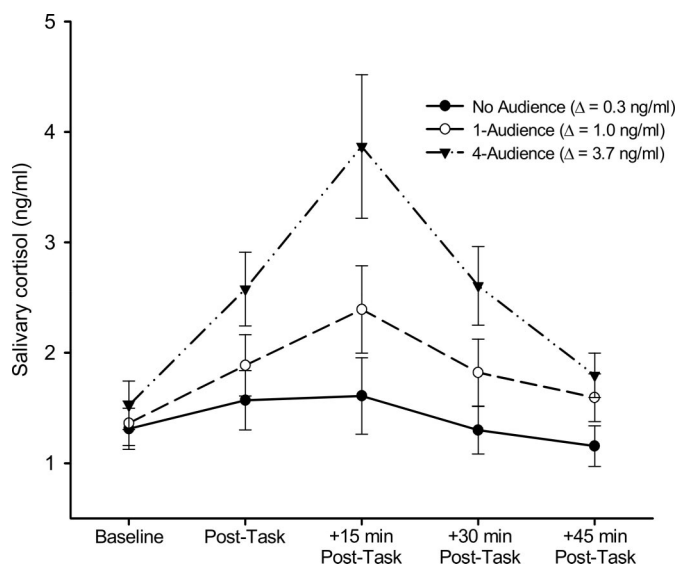
As shown in Figure 3, irrespective of condition, cortisol levels showed the expected peak at +15 minutes posttask and had largely returned to baseline levels at +45 minutes posttask (main-effect of time: $F(4,212) = 24.03$, $p < .001$, $\eta^2 = 0.312$). Separate analyses of each condition showed no significant cortisol change in the no-audience condition, $F(4,64) = 2.57$, $p = .085$, $\eta^2 = 0.139$, whereas there were main effects of time for both the 1-audience and 4-audience conditions, $F(4,76) = 8.84$, $p = .001$, $\eta^2 = 0.318$, and $F(4,72) = 15.55$, $p < .001$, $\eta^2 = 0.464$, respectively. Importantly, there was also a significant Time \times Condition interaction effect ($F(8,212) = 5.50$, $p = .001$, $\eta^2 = 0.172$). Table 2 presents the outcomes of pairwise comparisons of the cortisol responses during each condition. There were significantly larger cortisol changes in the 4-audience compared with the control condition and the 1-audience condition, whereas the 1-audience and control conditions were not significantly different (Table 2). Adjustment for gender, baseline cortisol, and BMI did not alter these outcomes.

Mediation Analyses

Attention then turned to whether the affective responses or autonomic reactivity predicted, i.e., mediated, the condition-dependent increases in cortisol. For mediation, it is necessary to show that a) the independent variable (task condition) affects the dependent variable (cortisol responses); b) the independent variable predicts the mediator (e.g., affective responses); c) the selected mediator predicts the dependent

TABLE 3. Univariate Statistical Analyses^a of Changes in Heart Rate, PEP, and RMSSD (Baseline, Task-1, Task-2, Recovery) and Cortisol (Baseline, Posttask, +15 Minutes, +30 Minutes, +45 Minutes Recovery)^b

	Heart Rate	PEP	RMSSD	Cortisol
Control vs. 1-audience	$F(3,111) = 3.75$, $p < .05$, $\eta^2 = 0.092$	$F(3,111) = 10.64$, $p < .001$, $\eta^2 = 0.223$	$F(3,111) = 6.30$, $p < .01$, $\eta^2 = 0.146$	$F(4,140) = 2.25$, $p = .107$, $\eta^2 = 0.060$
Control vs. 4-audience	$F(3,111) = 10.15$, $p < .001$, $\eta^2 = 0.215$	$F(3,111) = 20.70$, $p < .001$, $\eta^2 = 0.359$	$F(3,111) = 9.45$, $p < .001$, $\eta^2 = 0.203$	$F(4,136) = 8.32$, $p < .01$, $\eta^2 = 0.197$
1-Audience vs. 4-audience	$F(3,114) = 3.58$, $p < .05$, $\eta^2 = 0.086$	$F(3,114) = 4.22$, $p < .01$, $\eta^2 = 0.100$	$F(3,114) = 1.06$, $p = .369$, $\eta^2 = 0.027$	$F(4,148) = 4.27$, $p < .05$, $\eta^2 = 0.104$

^a Repeated-measures analysis of variance.^b Significant Time by Condition interactions are highlighted in bold. Results remained essentially unaltered after adjustment for gender and body mass index. PEP = preejection period; RMSSD = root mean square of successive differences.**Figure 3.** Mean \pm standard error of the mean salivary cortisol values in the no-audience (filled circles), 1-audience (open circles), and 4-audience (filled triangles) conditions (Δ = 15-minute posttask value – baseline value).

variable; and d) the association between the independent and the dependent variable is substantially attenuated after taking the putative mediator into account. In the preceding sections, we have shown a significant Time \times Condition interaction for cortisol, and importantly, that all the putative mediators (affective and autonomic responses) showed Time \times Condition interactions, satisfying the first and second condition for mediation.

Associations between candidate mediator variables and the dependent variable were tested using repeated-measures analysis of covariance (ANCOVA). To assess whether affective responses mediate the effect of condition on cortisol responses, changes in anxiety, shame/embarrassment, and negative affect were entered as covariates. ANCOVA was also used to test the extent to which mediators accounted for the association between the predictor (condition) and the dependent variable (cortisol response). If affective responses predict or mediate differential cortisol responses across conditions, this would result in a) a significant mediator by time interaction (i.e., the covariate would be significant), and b) an attenuation of the Time \times Condition interaction effect. However,

ANCOVA revealed that none of these covariates were significantly associated with cortisol responses; anxiety ($F(4,208) = 0.12$, $p = .851$, $\eta^2 = 0.002$), shame/embarrassment ($F(4,196) = 0.16$, $p = .816$, $\eta^2 = 0.003$), negative affect ($F(4,204) = 1.36$, $p = .260$, $\eta^2 = 0.026$). Unsurprisingly, the Time \times Condition interaction remained virtually unaltered when these parameters were entered as covariates. Together these results indicated that the condition effects on cortisol profile were unrelated to these affective responses.

To determine whether sympathetic cardiac responses during the tasks predicted subsequent cortisol responses, a similar mediation analyses was conducted, using change in PEP as a covariate in the ANCOVA. First, ANCOVA yielded a significant association between change in PEP and cortisol response ($F(4,208) = 4.13$, $p = .024$, $\eta^2 = 0.074$). Second, adjusting for PEP attenuated the significant cortisol Time \times Condition interaction to nonsignificance in conjunction with a substantial reduction in effect size: The variance in the cortisol response explained by condition fell from 17.2% (shown above) to 5.7% by adding PEP reactivity (Δ PEP) as a covariate ($F(8,208) = 1.86$, $p = .133$, $\eta^2 = 0.057$). This reduction was statistically significant ($F(8,212) = 4.10$; $p < .01$). Replicating these analyses, using cortisol area under curve as the dependent variable (as an alternative to using a repeated-measures approach), yielded virtually identical results. No evidence for mediation was found for HR variability (analyses not shown).

DISCUSSION

Social interaction and a need to belong are intrinsic to human existence, which makes it understandable that situations involving social transgression and threats to social standing are powerful stressors (49). The Social Self Preservation Theory (11,17) contends that HPA activation is a characteristic of such social stressors. The current results confirmed HPA activation in response to social evaluative threat. However, although cortisol activity clearly differentiated evaluative from nonevaluative task conditions, so too did HR, sympathetic cardiac activation (PEP), and vagal tone (RMSSD). Based on effect sizes, autonomic and cardiac reactions seemed equally or more sensitive than cortisol responses in differentiating social evaluative threat (SET) from non-SET.

RESPONSES TO SOCIAL EVALUATIVE THREAT

The current study manipulated levels of evaluative threat by increasing audience size (33). This enabled further examination of whether social evaluation determines the pattern, rather than the magnitude, of physiological reactivity. In keeping with previous findings (17), it was anticipated that the magnitude of cortisol reactivity, but not cardiac reactivity, would correlate positively with increasing audience size. The data did not support this prediction; cortisol, HR, and PEP all displayed comparable response gradients. For example, HR reactivity increased in a linear fashion from 9 beats/minute (no audience), to 14 beats/minute (1-audience), to 20 beats/minute (4-audience). RMSSD was the minor exception to this pattern; although vagal responses clearly differentiated evaluative from nonevaluative conditions, the difference in vagal withdrawal between the 1- and 4-audience conditions did not reach statistical significance. Taken together, however, it seems reasonable to conclude from the present data that social evaluation potentiates a general, rather than an HPA-specific, physiological reactivity.

Further support for a general reactivity-enhancing effect of SET was provided by mediation analyses, which indicated that the effects on HPA activation could not be separated from the effects on sympathetic activation. The finding that autonomic responses during the tasks predicted the subsequent elevation in cortisol replicates previous research (24,50–54), and is also consistent with evidence of extensive interaction between the two response systems (9,55–57). The present observations contrast, however, with the findings of Gruenewald et al. (17), which showed a relative insensitivity of cardiovascular measures to evaluative threat together with a selective cortisol reactivity. This discrepancy could have been due to confounding by movement and posture changes, which we aimed to minimize in the current study. A clear demonstration of the effects of such confounding is provided by Rohleder and colleagues (32). Their study showed that a control condition, imposing the mere physical activities of the TSST, elicited nearly similar changes in HR and heart rate variability as the full TSST protocol with the speech and mental arithmetic stressor (32). Thus, movement artifacts seem capable of partly masking the effects of the TSST stressors on the cardiovascular system.

The Social Self Preservation Theory is a rebuttal to assumptions based on Selye's generality theory which posits that all stressors will activate the HPA axis. The former partly rests on the outcome of a meta-analysis showing a clear difference in HPA activation between "regular" performance tasks (e.g., mental arithmetic) and performance tasks that include the additional element of social evaluation or uncontrollability (11). However, Selye's concept of stress, based on experiments that applied severe physical threats to animals, may also not generalize to the comparatively sedate human performance tasks for more fundamental physiological reasons: the activation of different physiological systems may require different intensities of provocation. That is, although mildly engaging problem-solving tasks readily perturb cardiac and autonomic activity, HPA activation seems to require more

provocative manipulations, such as evaluated speech tasks. Such tasks elicit higher levels of physiological activation in general² (19,23). A comparable elevated threshold for HPA activation is also observed during physical stressors like exercise (26). These observations lead us to tentatively propose an alternative "threshold activation" model of HPA reactivity during performance stressors. This threshold activation model postulates that some stressors, such as those without elements of social evaluation or uncontrollability, fail to induce a cortisol response simply because they are less likely to induce a level of activation sufficient to engage the HPA axis.

An implication of this threshold activation model is that provocative elements like social evaluation or uncontrollability engage the HPA axis not because of a unique psychological-physiological response association but by intensifying an otherwise moderately arousing task. Accordingly, evaluation can create a perception of response specificity when it is response intensity that is actually manipulated. An illustration of this phenomenon is provided by a study of Cacioppo and co-workers (53). In line with the extant literature (11,23,24), they observed that a simple performance task (mental arithmetic) does not create a significant average increase in cortisol. However, consistent with a threshold-activation model, further analyses of individual differences showed that the task did elicit cortisol release, but only in individuals exhibiting a strong cardiac autonomic activation. This finding is consistent with the idea that it is primarily the magnitude of physiological (e.g., autonomic) activation during a performance task that predicts whether the HPA axis becomes engaged.

Although the primary aim of our study was to assess the specificity of cortisol responses to social evaluative threat, our findings warrant a brief comment on the specificity of self-conscious emotions during such situations. It seems counter-intuitive that a context designed to elicit evaluation apprehension and which increases embarrassment and shame (emotions that reflect social threat) would not also increase apprehension and anxiety (49). However, such was the finding reported by Gruenewald and colleagues (17). In contrast, the current data showed that anxiety and shame/embarrassment increased in a parallel fashion. A possible explanation for this discrepancy could be that, in the Gruenewald study, participants in the social evaluation condition already reported elevated anxiety at baseline (compared with the nonevaluative condition). These baseline differences might have masked a differential effect of condition on anxiety. Another notable finding of the present study was that self-report measures revealed no differences between the 1- and 4-audience conditions, even though physiological responses clearly differentiated the conditions. This observation resembles that of other studies, which manipulated social context (58,59). Thus, it is possible that audience size drives physiological responses independent of affective mechanisms that were assessed here.

²Social evaluation adds several elements to a performance task thought to increase physiological reactivity, such as distraction, evaluation apprehension, and an incentive to perform well (19,63,64).

Several limitations should be noted. Like most research in this area, the present study was performed with university students, and replication of our findings in other populations is an important next step. Also, the group sizes were relatively small and the resulting lack in power requires a cautious interpretation of two null-findings: i.e., the lack of association between vagal reactivity and increasing audience size, and the absence of a significant correlation between shame/embarrassment and cortisol responses. A further limitation, shared with the study of Gruenewald et al. (17), is that the shame/embarrassment mood scale does not differentiate between these two distinct emotions, which may have different physiological correlates (60,61).

In summary, our findings were consistent with the general observation from social facilitation and social anxiety research that performance involving social evaluation elicits heightened physiological reactivity (19,20). We proposed a threshold activation model as an alternative explanation for the observation that social evaluative stressors characteristically elicit a cortisol response. This model is based on the observation that cortisol is less readily perturbed during psychological and physical stressors than cardiovascular and autonomic parameters. An illusion of response specificity may thus occur when a provocative element (e.g., lack of control, social evaluation) is incorporated into a less provoking challenge (e.g., performing arithmetic). We may add that this model does not imply that physiological responses to evaluative and nonevaluative stressors can simply be differentiated on a single dimension of activation or arousal (62). Our contention is merely that a threatening evaluative context seems to enhance broadly the reactivity of multiple physiological systems. It may be that this ability to elicit a robust generalized response explains why social stressors have been associated consistently with a range of health outcomes.

We thank Prof. John T. Cacioppo (University of Chicago) and Prof. Gary G. Berntson (Ohio State University) for comments on the rationale of the study and its interpretation. We also are very grateful for the help of Claire Sawyer, Gareth Tantram, Sarah Alderton, and Lewis Moore, who assisted with participant recruitment and experimental procedures (19,63,64).

REFERENCES

- Cacioppo JT, Tassinary LG. Inferring psychological significance from physiological signals. *Am Psychol* 1990;45:16–28.
- Stern RM, Sison CEE. Response patterning. In: Cacioppo JT, Tassinary LG, editors. *Principles of Psychophysiology: Physical, Social, and Inferential Elements*. Cambridge: Cambridge University Press; 1990.
- Kemeny ME. Psychobiological responses to social threat: evolution of a psychological model in psychoneuroimmunology. *Brain Behav Immun* 2009;23:1–9. Epub 2008 Sep 10.
- Schneiderman N, Ironson G, Siegel SD. Stress and health: psychological, behavioral, and biological determinants. *Annu Rev Clin Psychol* 2005;1:607–28.
- Taylor SE, Klein LC, Lewis BP, Gruenewald TL, Gurung RA, Updegraff JA. Biobehavioral responses to stress in females: tend-and-befriend, not fight-or-flight. *Psychol Rev* 2000;107:411–29.
- Weiner H. *Perturbing the Organism: The Biology of Stressful Experience*. Chicago: University of Chicago Press; 1992.
- Dienstbier RA. Arousal and physiological toughness: implications for mental and physical health. *Psychol Rev* 1989;96:84–100.
- Uchino BN, Cacioppo JT, Kiecolt-Glaser JK. The relationship between social support and physiological processes: a review with emphasis on underlying mechanisms and implications for health. *Psychol Bull* 1996;119:488–531.
- Sapolsky RM, Romero LM, Munck AU. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocr Rev* 2000;21:55–89.
- McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiol Rev* 2007;87:873–904.
- Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol Bull* 2004;130:355–91.
- Miller GE, Chen E, Zhou ES. If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychol Bull* 2007;133:25–45.
- Frankenhaeuser M. Challenge-control interaction as reflected in sympathetic-adrenal and pituitary-adrenal activity: comparison between the sexes. *Scand J Psychol* 1982;Suppl 1:158–64.
- Henry JP. Neuroendocrine patterns of emotional response. In: Plutchick R, Kellerman H, editors. *Emotion: Theory, Research and Experiences*. San Diego: Academic Press; 1986.
- Schommer NC, Hellhammer DH, Kirschbaum C. Dissociation between reactivity of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medullary system to repeated psychosocial stress. *Psychosom Med* 2003;65:450–60.
- Dickerson SS, Gruenewald TL, Kemeny ME. When the social self is threatened: shame, physiology, and health. *J Pers* 2004;72:1191–216.
- Gruenewald TL, Kemeny ME, Aziz N, Fahey JL. Acute threat to the social self: shame, social self-esteem, and cortisol activity. *Psychosom Med* 2004;66:915–24.
- Kirschbaum C, Pirke KM, Hellhammer DH. The ‘Trier Social Stress Test’—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology* 1993;28:76–81.
- Cacioppo JT, Rourke PA, Marshall-Goodell BS, Tassinary LG, Baron RS. Rudimentary physiological effects of mere observation. *Psychophysiology* 1990;27:177–86.
- Wright RA, Tunstall AM, Williams BJ, Goodwin JS, Harmon-Jones E. Social evaluation and cardiovascular response: an active coping approach. *J Pers Soc Psychol* 1995;69:530–43.
- Smith TW, Nealey JB, Kircher JC, Limon JP. Social determinants of cardiovascular reactivity: effects of incentive to exert influence and evaluative threat. *Psychophysiology* 1997;34:65–73.
- Kamarck TW, Annunzio B, Amateau LM. Affiliation moderates the effects of social threat on stress-related cardiovascular responses: boundary conditions for a laboratory model of social support. *Psychosom Med* 1995;57:183–94.
- Linden W, Rutledge T, Con A. A case for the usefulness of laboratory social stressors. *Ann Behav Med* 1998;20:310–6.
- Al’Absi M, Bongard S, Buchanan T, Pincomb GA, Licinio J, Lovallo WR. Cardiovascular and neuroendocrine adjustment to public speaking and mental arithmetic stressors. *Psychophysiology* 1997;34:266–75.
- Ewart CK, Kolodner KB. Social competence interview for assessing physiological reactivity in adolescents. *Psychosom Med* 1991;53:289–304.
- Luger A, Deuster PA, Gold PW, Loriaux DL, Chrousos GP. Hormonal responses to the stress of exercise. *Adv Exp Med Biol* 1988;245:273–80.
- Berntson GG, Uchino BN, Cacioppo JT. Origins of baseline variance and the Law of Initial Values. *Psychophysiology* 1994;31:204–10.
- Veldhuijzen van Zanten JJ, Ring C, Burns VE, Edwards KM, Drayson M, Carroll D. Mental stress-induced hemoconcentration: sex differences and mechanisms. *Psychophysiology* 2004;41:541–51.
- Tulen JH, Boomsma F, Man in ’t Veld AJ. Cardiovascular control and plasma catecholamines during rest and mental stress: effects of posture. *Clin Sci (Lond)* 1999;96:567–76.
- Obrist PA. *Cardiovascular Psychophysiology: A Perspective*. New York: Plenum Press; 1981.
- Houtveen JH, Groot PF, Geus EJ. Effects of variation in posture and respiration on RSA and pre-ejection period. *Psychophysiology* 2005;42:713–9.
- Rohleder N, Wolf JM, Maldonado EF, Kirschbaum C. The psychosocial stress-induced increase in salivary alpha-amylase is independent of saliva flow rate. *Psychophysiology* 2006;43:645–52.

RESPONSES TO SOCIAL EVALUATIVE THREAT

33. Knowles ES. Social physics and the effects of others: tests of the effects of audience size and distance on social judgements and behavior. *J Pers Soc Psychol* 1983;45:1263–79.
34. Kirschbaum C, Kudielka BM, Gaab J, Schommer NC, Hellhammer DH. Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosom Med* 1999;61:154–62.
35. Bosch JA, de Geus EJ, Veerman EC, Hoogstraten J, Nieuw Amerongen AV. Innate secretory immunity in response to laboratory stressors that evoke distinct patterns of cardiac autonomic activity. *Psychosom Med* 2003;65:245–58.
36. Bosch JA, Berntson GG, Cacioppo JT, Marucha PT. Differential mobilization of functionally distinct natural killer subsets during acute psychologic stress. *Psychosom Med* 2005;67:366–75.
37. Saab PG, Matthews KA, Stoney CM, McDonald RH. Premenopausal and postmenopausal women differ in their cardiovascular and neuroendocrine responses to behavioral stressors. *Psychophysiology* 1989;26:270–80.
38. van Eck MM, Nicolson NA, Berkhof H, Sulon J. Individual differences in cortisol responses to a laboratory speech task and their relationship to responses to stressful daily events. *Biol Psychol* 1996;43:69–84.
39. Sarason IG, Stoops R. Test anxiety and the passage of time. *J Consult Clin Psychol* 1978;46:102–9.
40. Leary MR. Social anxiousness: the construct and its measurement. *J Pers Assess* 1983;47:66–75.
41. Heatherton TF, Polivy J, Herman CP. Restraint, weight loss, and variability of body weight. *J Abnorm Psychol* 1991;100:78–83.
42. Derogatis LR, Yevzeroff H, Wittelsberger B. Social class, psychological disorder, and the nature of the psychopathologic indicator. *J Consult Clin Psychol* 1975;43:183–91.
43. Berntson GG, Cacioppo JT, Quigley KS. Cardiac psychophysiology and autonomic space in humans: empirical perspectives and conceptual implications. *Psychol Bull* 1993;114:296–322.
44. Bosch JA, de Geus EJ, Kelder A, Veerman EC, Hoogstraten J, Amerongen AV. Differential effects of active versus passive coping on secretory immunity. *Psychophysiology* 2001;38:836–46.
45. Willemsen GH, De Geus EJ, Klaver CH, Van Doornen LJ, Carroll D. Ambulatory monitoring of the impedance cardiogram. *Psychophysiology* 1996;33:184–93.
46. Bosch JA, Berntson GG, Cacioppo JT, Dhabhar FS, Marucha PT. Acute stress evokes selective mobilization of T cells that differ in chemokine receptor expression: a potential pathway linking immunologic reactivity to cardiovascular disease. *Brain Behav Immun* 2003;17:251–9.
47. Berntson GG, Cacioppo JT, Binkley PF, Uchino BN, Quigley KS, Fieldstone A. Autonomic cardiac control. III. Psychological stress and cardiac response in autonomic space as revealed by pharmacological blockades. *Psychophysiology* 1994;31:599–608.
48. Cacioppo JT, Berntson GG, Binkley PF, Quigley KS, Uchino BN, Fieldstone A. Autonomic cardiac control. II. Noninvasive indices and basal response as revealed by autonomic blockades. *Psychophysiology* 1994;31:586–98.
49. Baumeister RF, Leary MR. The need to belong: desire for interpersonal attachments as a fundamental human motivation. *Psychol Bull* 1995;117:497–529.
50. Uchino BN, Cacioppo JT, Malarkey W, Glaser R. Individual differences in cardiac sympathetic control predict endocrine and immune responses to acute psychological stress. *J Pers Soc Psychol* 1995;69:736–43.
51. Sgoutas-Emch SA, Cacioppo JT, Uchino BN, Malarkey W, Pearl D, Kiecolt-Glaser JK, Glaser R. The effects of an acute psychological stressor on cardiovascular, endocrine, and cellular immune response: a prospective study of individuals high and low in heart rate reactivity. *Psychophysiology* 1994;31:264–71.
52. Sgoifo A, Braglia F, Costoli T, Musso E, Meerlo P, Ceresini G, Troisi A. Cardiac autonomic reactivity and salivary cortisol in men and women exposed to social stressors: relationship with individual ethological profile. *Neurosci Biobehav Rev* 2003;27:179–88.
53. Cacioppo JT, Malarkey WB, Kiecolt-Glaser JK, Uchino BN, Sgoutas-Emch SA, Sheridan JF, Berntson GG, Glaser R. Heterogeneity in neuroendocrine and immune responses to brief psychological stressors as a function of autonomic cardiac activation. *Psychosom Med* 1995;57:154–64.
54. Doussard-Roosevelt JA, Montgomery LA, Porges SW. Short-term stability of physiological measures in kindergarten children: respiratory sinus arrhythmia, heart period, and cortisol. *Dev Psychobiol* 2003;43:230–42.
55. Habib KE, Gold PW, Chrousos GP. Neuroendocrinology of stress. *Endocrinol Metab Clin North Am* 2001;30:695–728; vii–viii.
56. Cacioppo JT. Social neuroscience: autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology* 1994;31:113–28.
57. Wang J, Rao H, Wetmore GS, Furlan PM, Korzykowski M, Dinges DF, Detre JA. Perfusion functional MRI reveals cerebral blood flow pattern under psychological stress. *Proc Natl Acad Sci U S A* 2005;102:17804–9.
58. Gerin W, Pieper C, Levy R, Pickering TG. Social support in social interaction: a moderator of cardiovascular reactivity. *Psychosom Med* 1992;54:324–36.
59. Kamarck TW, Manuck SB, Jennings JR. Social support reduces cardiovascular reactivity to psychological challenge: a laboratory model. *Psychosom Med* 1990;52:42–58.
60. Leary MR. Motivational and emotional aspects of the self. *Annu Rev Psychol* 2007;58:317–44.
61. Drummond PD, Back K, Harrison J, Helgadottir FD, Lange B, Lee C, Leavy K, Novatscou C, Orner A, Pham H, Prance J, Radford D, Wheatley L. Blushing during social interactions in people with a fear of blushing. *Behav Res Ther* 2007;45:1601–8.
62. Berntson GG. Reasoning about brains. In: Cacioppo JT, Visser PS, Pickett CL, editors. *Social Neuroscience: People Thinking About People*. Cambridge, MA: MIT Press; 2006.
63. Baron RS. Distraction-conflict theory: progress and problems. *Adv Exp Soc Psychol* 1986;19:1–40.
64. Wright RA, Killebrew K, Pimpalpure D. Cardiovascular incentive effects where a challenge is unfixed: demonstrations involving social evaluation, evaluator status, and monetary reward. *Psychophysiology* 2002;39:188–97.